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Acute Methaemoglobinaemia following ingestion of amyl nitrite

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Abstract

Presentation
The patient presented with an acute severe desaturation to 74% on room air. Initial management included 15 litres of oxygen via non-rebreather mask and continuous monitoring. Despite this, he remained markedly cyanosed and tachycardic.

Diagnosis
Arterial blood gas (ABG) analysis revealed methaemoglobinemia and a characteristic dark brown discoloration of the blood. *Figure 1.* Methylene Blue was considered, but conservative management was continued due to clinical improvement.

Treatment
After one hour, cyanosis and tachycardia had improved, and an ABG showed decreased methaemoglobinemia. A fourth ABG at 4 hours showed a methaemoglobinaemia less than 3%.

Discussion
It was later discovered that the patient had ingested a bottle of “poppers”, a commonly abused drug containing amyl nitrite.

Introduction

In this case report we demonstrate a conservative approach to the management of severe methaemoglobinaemia secondary to the ingestion of “poppers” a common street drug containing amyl nitrite. Methaemoglobinaemia is a rare blood disorder associated with oxidisation of divalent ferrous iron [Fe^{2+}] in haemoglobin (Hb) to ferric iron [Fe^{3+}] in methaemoglobin (MetHb), giving the blood a characteristic ‘chocolate brown colour’, and rendering it unable to bind oxygen effectively[1]. Patients can acquire methaemoglobinaemia after exposure to certain drugs, such as local anaesthetics, nitrites, and antibiotics[2]. Its´ treatment usually relies on methylene blue infusion. The main constituent of ‘poppers’, a commonly abused drug is amyl nitrite.

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Case Presentation

A 39-year-old man with a known history of substance misuse presented on the postoperative ward, with severe desaturation (74%) and marked cyanosis. Other clinical findings included a heart rate of 143 beats per minute, with no associated signs of shock or cardiac failure. The patient reported feeling dizzy and having a headache. Initial management included administration of 15L oxygen via a non-rebreather mask. Electrocardiogram (ECG), chest X-ray and arterial blood gas (ABG) analysis were obtained. Vital signs were monitored. A full set of bloods and d-dimer were also sent. The patient's oxygen saturation remained low (maximum 84%), his other vitals were stable. The ECG and chest X-ray were unremarkable. Electrolytes, D-dimer, renal function, and the haemogram were normal. ABG analysis revealed a partial pressure of oxygen (pO2) 28.67mmHg, and an elevated methaemoglobin level of 37.6%. The arterial blood obtained was dark brown, consistent with methaemoglobinaemia (*Figure 1).* This patient continued to receive 15 litres of oxygen via a non-rebreather mask and was moved to an observation room where his vitals could be monitored. We observed an improvement in saturations, cyanosis and tachycardia, allowing progressive weaning from supplemental oxygen. A repeat ABG taken 1 hour after the initial presentation showed methaemoglobin level of 26%. Intravenous Methylene Blue infusion was considered but given his clinical improvement and down trending methaemoglobin levels, the decision was made to continue conservative management. Repeat ABGs were taken every hour and by the 4th ABG, the methaemoglobin level was below 3%.

Discussion

Methaemoglobinaemia can occur secondary to exposure to various substances, including drugs such as local anaesthetics, nitrites and antibiotics. In this case, the aetiology of methaemoglobinaemia was not immediately apparent. It was later discovered that the patient had ingested a small bottle of the illlicit drug 'poppers', in which the main constituent is amyl nitrite, a known cause of methaemoglobinaemia. The half-life for amyl nitrite is less than one hour[3]. Haemoglobin normally contains ferrous iron [Fe^{2+}] which has a high affinity for oxygen, allowing effective oxygen transport to tissues. In methaemoglobinemia iron is oxidised to the ferric state [Fe^{3+}], impairing its affinity for oxygen. This decreases oxygen delivery to the tissues. The presence of ferric iron in haemoglobin also increased the affinity of ferrous iron for oxygen, resulting in a left shift in the oxy-haemoglobin dissociation curve, impairing oxygen release at a tissue level. Prompt recognition and determining the aetiology of methaemoglobinaemia is essential, as delayed diagnosis and treatment can lead to serious complications. Treatment options include oxygen therapy, methylene blue administration, high dose vitamin C and red blood cell transfusion. These therapies work by reducing ferric iron in methaemoglobin to ferrous iron,

improving haemoglobins affinity for oxygen. The patient was managed with oxygen therapy and gradually improved. He did not require methylene blue administration. This case highlights that methaemoglobinaemia should be considered as a differential in an acute desaturation case. Timely recognition is crucial for optimal patient outcomes. Further research is needed to develop clear management guidelines for methaemoglobineamia.

Declarations of Conflicts of Interest:
None declared.

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Figure 1